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保留比值受损肺功能人群的临床研究进展

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摘　要

保留比值受损肺功能(PRISm)作为一种重要的慢性阻塞性肺疾病(COPD)前期表型，近年引起学界高度关注。该人群虽未达COPD肺功能诊断标准(FEV1/FVC ≥ 0.7)，但存在肺功能指标(FEV1和/或FVC < 80%预计值)损害、影像学异常，且不良预后风险显著增高。本文系统梳理近期PRISm相关文献，聚焦其流行病学特征、风险因素、临床分型、临床特征、检查手段，旨在为PRISm人群的早期识别、干预策略制定及未来研究方向提供依据。

关键词

保留比值受损肺功能，慢性阻塞性肺疾病，肺功能检查

Progress in Preserved Ratio Impaired Spirometry

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Abstract

Preserved ratio impaired spirometry (PRISm), recognized as a significant pre-COPD phenotype, has attracted considerable research interest. Individuals with PRISm do not fulfill diagnostic criteria for COPD yet demonstrate impaired lung function, frequent radiological abnormalities, and elevated risks of adverse outcomes. This review synthesizes current evidence on PRISm, encompassing its

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epidemiology, risk determinants, clinical typing, clinical characteristics, diagnostic approaches, with the goal of informing early detection, intervention, and clinical investigation for this population.

Keywords

Preserved Ratio Impaired Spirometry, Chronic Obstructive Pulmonary Disease, Pulmonary Function Test

1. 引言

慢性阻塞性肺疾病(COPD)是一种以持续性、进行性气流受限为特征的异质性呼吸系统疾病，源于气道和(或)肺泡结构异常，其高发病率、死亡率及沉重疾病负担严重影响患者生活质量并消耗大量医疗资源[1] [2]。虽然 COPD 尚无法根治，但早期识别肺功能受损或 COPD 前期状态对于症状控制与不良结局预防至关重要。2024 年 GOLD 指南明确定义 PRISm 为：支气管舒张剂后 FEV1/FVC ≥ 0.7，同时 FEV1＜80%预计值和(或) FVC < 80%预计值，旨在识别具有慢性气流阻塞风险的早期个体。尽管未达 COPD 诊断阈值，PRISm 人群常报告慢性呼吸道症状，且心血管疾病、糖尿病等共病风险显著增加[3]-[5]。研究一致表明，相较于肺功能正常者，PRISm 人群进展为 COPD 的风险、心血管相关死亡率及全因死亡率均显著增高[6]。鉴于其过渡性特征及潜在不良预后，PRISm 不宜简单视为 COPD 早期阶段，亟需加强关注以推动早期识别与干预。国内对 PRISm 研究尚显不足。本文综述国内外最新成果，围绕 PRISm 的流行病学特征、风险因素、临床分型、临床特征、检查手段进行阐述，以期为其诊疗提供参考。

2. PRISm 的流行病学特征

全球 PRISm 患病率介于 4.7%~25.2% [7]。BOLD 研究(涵盖 14 国)显示 ≥ 40 岁人群 PRISm 患病率达 14.2% [3]；荷兰 Rotterdam 研究(≥45 岁)患病率为 7.1% [6]；英国 Biobank 研究(2006~2010)患病率为 11% [8]。亚洲地区，日本 OCEAN 研究显示患病率 16.7% [9]，韩国全国调查(2010~2019)为 10.4% [10]。中国成人肺部健康研究(CPH)年龄标化患病率为 5.5% [11]，而早期 BOLD 研究(2002 年)显示广州数据高达 23.6% [6]。兰勇兵等人基于中国慢性病前瞻性研究(CKB)发现，≥40 岁人群中 PRISm 患病率达 24.8%，农村(25.4%)略高于城市(24.3%)，地区差异显著(甘肃最高 56.0%，河南最低 15.4%) [12]。中国肺健康研究揭示高原地区(平均海拔 3507 m)患病率为 10.05% [11]。上述数据凸显 PRISm 患病率存在显著地域及人群差异，可能与种族、地理环境、选用肺功能预计值不同等因素相关。值得注意的是，亚洲尤其我国 PRISm 负担突出，提示国内存在庞大患病人群，迫切需要进行全国性多中心大样本流行病学调查，以明确实际患病率及疾病特征，指导防治工作。性别分布上，多项研究[3] [9] [13]表明 PRISm 人群以女性为主，挑战了肺功能下降主要见于男性吸烟者的传统认知。Mannino D.M.等人的研究[3]显示男性患病为 11.7% (546/4664)，女性达 16.4% (836/5098)。年龄方面，≥40 岁人群风险显著增高，但年轻群体(尤其合并哮喘或肥胖者)亦存在。澳大利亚 BOLD 研究[11]发现，相比肺功能正常或气流受限者，PRISm 受试者更年轻、女性比例更高、非吸烟者更多，且更可能来自社会经济弱势地区或为肥胖个体。

3. PRISm 的潜在危险因素

目前研究已识别多种 PRISm 高危因素，包括：高龄[14]、女性[6] [8] [15]、吸烟[15]、异常体质量指数(BMI 过高或过低) [6] [8]、职业粉尘暴露[16]、生物燃料暴露[17]、肺生长发育异常[18]、哮喘病史[19]、糖尿病[4]、睡眠呼吸暂停综合征[20]、小气道功能障碍及总肺活量降低[21]、高血压[3]、陈旧性肺结核[4]、低社会经济地位及低教育水平[6] [8]。

新近研究提示环境重金属暴露亦是风险因素。美国研究[22]显示血清镉水平升高与 PRISm 风险增高及肺功能(尤其 FEV1)下降显著相关。我国台湾横断面研究发现[23]，血清铟异常组 PRISm 患病率(22.7%)显著高于正常组(7.6%)。

大量研究表明同样与代谢相关：膳食脂肪酸(如饱和脂肪酸、α-亚麻酸)摄入与 PRISm 风险呈负相关，机器学习模型提示丁酸(SFA 4:0)为关键亚类[24]。美国研究[25]显示葡萄糖处理率(eGDR)降低与 PRISm 患病率升高显著相关，提示胰岛素抵抗和代谢功能障碍的作用，eGDR 或可作为评估风险(尤其女性和老年人)的生物标志物。NHANES 数据(2007~2012) [26]表明血清非高密度脂蛋白胆固醇/高密度脂蛋白胆固醇比值(NHHR)与 PRISm 正相关且与肺功能下降显著关联。佟琳等人的研究[27]发现维生素 D 水平与 PRISm 风险呈负相关(OR: 0.989, 95%CI: 0.984~0.994)，胆红素可能介导此保护效应，提示维持充足维生素 D 水平或有益于肺健康。Jesus 等人的研究[28]揭示 PRISm 及 COPD 患者血清脱氢表雄酮(DHEA)及其硫酸盐(DHEA-S)水平降低，且二者与 FEV1、FVC 呈正相关。

4. PRISm 的异质性及临床分型

研究显示 PRISm 表型具有动态演变性：少数可逆转为正常肺功能，部分维持 PRISm 状态，部分进展为 COPD。鹿特丹研究[6]基于 PRISm 纵向转归将 PRISm 分为三个亚组：进展至 COPD 组、高心血管负担及早期死亡组、持续性 PRISm 组。4.5 年随访期内，5.7% PRISm 受试者肺功能转归正常，49.4%进展为 COPD；9.3 年随访期间，PRISm 组死亡率 18.7%，COPD 组 20.8%，正常对照组 10.3%。英国一项老龄化纵向研究[29]根据基线肺功能特征将 PRISm 分为轻度 PRISm (FEV1 或 FVC 减少)和重度 PRISm 亚型(FEV1 和 FVC 均减少)，研究显示重度 PRISm 较轻度 PRISm 年龄更大、教育程度更低、目前吸烟的比例更高；同时，重度 PRISm 相较于轻度 PRISm 具有更严重的呼吸道症状(如咳痰、喘息、呼吸困难、慢性支气管炎及肺气肿)，FVC 平均值更低，死亡风险更高，进展为 GOLD2~4 级 COPD 的概率更高。亦有研究[30]根据是否存在限制性肺量异常将 PRISm 分为：非限制性 PRISm (FEV1/FVC ≥ 0.7, FEV1 < 80%, FVC ≥ 80%)和限制性 PRISm (FEV1/FVC ≥ 0.7, FEV1 < 80%, FVC < 80%)；前者在传统标准中可归类正常，但与气流阻塞风险增高相关；多因素分析显示哮喘史和吸烟史与非限制性 PRISm 独立相关，而女性、高龄和高 BMI 是限制性 PRISm 的独立危险因素。

5. PRISm 的临床特征与合并症

PRISm 人群常呈现与 COPD 相似的呼吸道症状[26] [8]，如慢性咳嗽、咳痰、喘息及呼吸困难。然而，相较于 COPD，PRISm 人群呼吸困难风险更高，六分钟步行距离更短，支气管扩张剂使用前后肺功能值更低，且接受呼吸治疗比例较低[15]。

PRISm 与多种系统性疾病共病密切相关，包括高龄[14]、肥胖[6] [8] [14]、高血压[3] [5]、糖尿病[3] [4]、心力衰竭、冠心病[3]、脑卒中、衰弱、痴呆等。韩国的一项研究[31]证实 PRISm 个体上述共病发生率显著高于其他队列。与肺功能正常者相比[3]，PRISm 人群糖尿病(12.2% vs 4.6%)、心脏病(15.0% vs 7.7%)、高血压(38.8% vs 22.8%)患病率显著增高。最新全基因组关联研究(GWAS) [5]揭示 PRISm 与代谢及心血管疾病存在遗传关联，发现 22 个信号与糖尿病相关性状、7 个与血压变化相关。英国生物样本库研究[32]

还显示 PRISm 与痛风风险增加显著相关，部分由尿酸水平、炎症标志物及免疫细胞计数介导。

6. PRISm 的检查方法

PRISm 主要通过肺功能检测(pulmonary function test, PFT)发现，其 GOLD 诊断标准依赖 PFT 结果。然而，PFT 准确性受患者状态及配合度影响。近期研究[33]强调胸部 CT 在 PRISm 评估中的价值，该人群常存在气道壁增厚、肺气肿(小叶中心型或间隔旁型)、气体陷闭等结构性改变。定量 CT 参数(如肺气肿指数 EI、空气潴留指数 ATI)可客观量化肺结构损伤，辅助风险分层。有研究表明[34]，极低的 CT 评估肺活量/总肺容量(FVC/TLC_{CT})比值(最低四分位数)与总体呼吸系统急性加重风险增加显著相关(IRR = 1.65; 95% CI [1.07~2.54])，PRISm 患者 FVC/TLC_{CT} 降低与症状增多、影像学肺气肿/气体滞留、急性加重及进展为 COPD 相关。

电阻抗断层扫描(EIT)作为一种无创成像技术，可评估通气时空分布。FEV1/FVC < 0.7 区域的存在可能反映局部生理异常(如气道水肿/痉挛)和/或结构性病变(如肺气肿)、空气滞留、过度充气及弥散功能降低。研究表明[35]，相较于传统肺量计，EIT 在评估有呼吸道症状(含 COPD 前期及 PRISm)门诊患者肺区域功能方面更具优势。

呼气挥发性有机化合物(VOC)分析是呼吸疾病无创鉴别诊断的新兴方法。研究发现[36]特定 VOC (如二甲苯、M-p-二甲苯、丙酮)水平变化与 PRISm 潜在相关，其中丙酮降低可能预示呼气流量减少。血清标志物如维生素 D [27]、DHEA(-S) [28]、炎症标志物(CRP、IL-6、血 EOS 计数)、代谢标志物(胰岛素、脂质谱、尿酸[32])、重金属(镉[22]、铟[23])等，可能反映特定亚型的驱动机制(如代谢、炎症、环境暴露等)。

脉冲振荡法(IOS)用于无创评估气道阻力及呼吸力学。研究发现[37]，相较于使用正常下限(LLN)标准(FEV1/FVC ≥ LLN 且 FEV1 < LLN)，采用固定比率(FR)标准(FEV1/FVC ≥ 0.7 且 FEV1 < 80%预计值)定义的 PRISm 患病率更高(10.0% vs. 4.2%)。IOS 参数 R5~R20 ≥ 120%预计值在识别 PRISm 时展现出最高的灵敏度和特异性。

7. 讨论及展望

尽管 PRISm 研究近年来取得了显著进展，但仍面临多方面的挑战。如诊断标准尚未统一且存在适用性争议：PRISm 定义是使用固定比值(FR)还是基于健康人群的正常下限(LLN)标准，FR 因其简便性被 GOLD 指南采用，但可能导致过度诊断并增加人群异质性(研究显示其患病率显著高于 LLN 标准[37])，而 LLN 标准虽更生理性却可能遗漏有临床意义者；此外，全球 PRISm 患病率差异较大，除不同种族、地区影响外，研究选择的不同肺功能预计值公式(大多数研究选用 FEV1/FVC ≥ 0.7 且 FEV1 < 80%预计值，并未要求 FVC < 80%预计值)直接导致了研究结果的显著异质性。同时阻碍进一步 PRISm 的深入研究是病理生理机制碎片化：现有研究虽提示 PRISm 与小气道病变、肺气肿、肥胖限制效应、代谢紊乱、炎症、环境暴露及发育异常等多种因素相关，但这些因素如何相互作用、主导特定亚型、驱动疾病进展，仍缺乏系统性认识，且多数证据来自横断面或回顾性研究，难以明确因果关系和构建完整的机制链条。基于现有数据，我们可以看出 PRISm 的病理生理主要有三个方面：肺部结构性及功能性易损基础(由遗传因素如 GWAS 发现的信号[5]、早期生命事件及肺生长发育异常导致的终末气道或肺泡结构缺陷[18]、小气道固有脆弱性、肺实质弹性回缩力减弱倾向等)、局部肺部损伤与炎症(由环境暴露、感染或局部免疫失调导致小气道炎症、杯状细胞增生或粘液分泌增加、气道壁重塑、肺泡壁破坏等)、系统性炎症与代谢失调(肥胖[3] [8]、代谢综合征[15]-[17]、心血管疾病[3]、衰老或全身性疾病[3] [4] [20]产生系统性炎症介质溢出至肺部，加剧局部炎症和损伤；同时，肺部局部炎症也可加剧系统性炎症，形成恶性循环)。

作为 COPD 前期的重要表型，PRISm 的高患病率、动态演变性及不良预后对公共卫生构成严峻挑战，

其早期识别与干预对 COPD 防控意义重大。现有研究初步阐明了其流行病学、风险因素、临床特点及检查方法，但在病理生理机制、精准诊断工具及有效干预策略等方面仍存不足。未来需强化多学科交叉研究，推动精准诊疗技术转化应用，以期实现对 PRISm 人群的早期有效干预，最终改善其临床预后。

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